Clinical study of shock in children with special reference to prognostic determinant at teaching hospital ,Bangalore.

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Abstract

Background: Limited information from developing countries regarding early prognostic determinants of shock in children prompted this study . Clinical and laboratory parameters help inassessing severity, patient management, prognostication, and optimal utilization of resources resulting in improved outcome at early stage.

Objectives: To find out association of various clinical and monitoring parameters of shock with outcome.

Methods: This was a prospective observational studyconducted from October 2005 to September 2006 at Indira Gandhi Institute of Child Health (IGICH) Bangalore. Hundred children above1 month upto 16 years admitted with shock in the pediatric intensive care unit (PICU) were selected . Parameters like heart rate, respiratory rate, blood pressure, capillary refilling time(CRT), Glasgow coma scale (GCS) , urine output, Core-peripheral temperature-gradient (C-PTG), oxygen saturation (SpO_2) were monitered. Clinical and laboratory parameters were compared between survivors and nonsurvivors

Results: Common etiology was septic shock (48%), followed by hypovolemic (28%) and cardiogenic shock (23%) ; highest mortality was observed in septic shock (65.5%) followed by cardiogenic shock(31%). Least mortality was seen in hypovolemic and anaphylactic shock. Parameters which remained abnormal inspite of treatment like persistent tachycardia, low blood pressure, prolonged CRT, high C-PTG, low GCS , decreased urine output, thrombocytopenia, high creatinine levels, low PaO_2, high PCO_2, lowSPO_2 were associated with increased mortality.

Conclusion: In survivors the trend was towards normalization of clinical parameters in first 24-48 hours of admission whereas they tended tobepersistantly abnormal in non-survivors.

Keywords:CRT, C-PTG, GCS ,PaO_2, SpO_2 .

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Introduction

Shock or circulatory failure is an acute syndrome characterized by inadequate circulatory perfusion of tissues to meet the metabolic demands of vital organs and, if prolonged, leads to multiple organ failure and death [1,2]. Shock is one of the commonest pediatric emergencies [3]. Unlike adults, hypotension is a very late feature of shock in children. As the child's condition worsens, the clinical presentation of shockof different etiology become similar, and nullify any aetiological differences. Regardless of the type ofshock, the final common pathway is inadequate tissue perfusion and oxygen supply to meet cellular demands. Delayed recognition and treatment result in progression from compensated reversible shock to uncompensated irreversible shock with widespread multiple system organ failure to death[4]. These children with shock are often referred to tertiary care facility for admission and management. The time lapse between the onset of this state , the time of admission and initiation of resuscitative measures is a great factor in determining the outcome[5]. These children are treated in a pediatric intensive care setup where constant observation and vigil with appropriate monitoring of various clinical parameters and laboratory parameters will determine and modify the therapeutic intervention which in turn will determine the outcome.

The mortality rate is extremely high in septic shock even in developed countrieswhere as the outcome in shock states secondary to envenomation is extremely gratifying[3]. Extremely gratifying outcome at one end and extreme mortality at the other prompted this study to be undertaken so as to find out the occurrence of this problem among pediatric admissions, the various causes contributing to them and to assess the outcome in relation to the various clinical and monitoring parameters.

Aims and Objectives

1. To study the occurrence of shock states and categorize it based on etiology .
2. To find out association of various clinical and monitoring parameters of shock with outcome.

Material and Methods

This hospital based prospective observational study was carried out in Pediatric Intensive Care Unit(PICU) at Indira Gandhi Institute of Child Health at Bangalore (Tertiary care paed. Center) from October 2005 to September 2006 .This study was approved by Institutional Ethics Committee, prior to commencement. Informed and written consent of parent and guardian was taken before including the child in the study**.** Therewere total 784 children more than 1 month and upto 16 years admitted to PICU **.**Neonates and Children who die within one hour after admission and patients in terminal state of cardiorespiratory failure were excluded. After excluding 100(12.7%) consecutive PICU children with a clinical diagnosis of shock were selected for the study and their clinical and investigational parameters were studied and compared between survivors and non-survivors . Once the patient was presented to the emergency room the relevant data regardingdetailed history was noted in the proforma which was pre-structured , pre-tested. Detailedclinicalexamination regarding shock assessment was done quickly while instituting appropriate treatment.The patients were monitored andrecordedperiodicallyfor the following parametersduring the hospital stay from the time of presentation at 0, 12, 24 and 48 hours after admission : Heart rate, Blood pressure, Respiratory rate , Capillary Refill Time (CRT), Core-peripheral temperature gradient (C-PTG), Glasgow Coma Scale (GCS), Oxygen saturation (SpO_2) and Urine output. Consciousness was assessed using modified GCS for infants and children. Heart rate was obtained from the multichannel monitoring. Also the pulse

was felt and its character assessed, as well as blood pressure recording was obtained non-invasively. Respiratory rate was counted and recorded. Capillary refill time was recorded in the following manner the upper limb was raised slightly above the level of the heart and firm pressure was applied by the clinician's index finger and thumb to the distal phalanx of the patient's index finger for five seconds. The finger was then released and the time taken for the palmar pulp to return to its previous color was recorded. Times were measured to the nearest second by a wristwatch. Core temperature was measured rectally and peripheral temperature taken on the distal aspect that was not overtly ischemic. SpO_2 was measured by pulse oximetry.

All the patients were catheterized and the urine output was measured. Arterial blood gas analysis was done and pH, partial pressure of carbon dioxide (PCO_2) and partial pressure of oxygen (PaO_2) values were noted. The investigations was done at the time of admission. Subsequently blood was taken for hematological studies (platelet count) and biochemical measurements (blood urea, serum creatinine, serum bilirubin, aspartate aminotransferase (AST), alanine aminotransferse (ALT) and prothrombin time for all the patients. Other relevant investigations were done depending on the individual case. Central venous pressure was monitored in cases where it was it was needed. Shock as a clinical state was dignosed in which the recorded blood pressure was <2 standard deviations below the mean for age and/or a state in which at least three of the following criteria for decreased perfusion were identified. Decreased peripheral pulses; Mottled or coolextremities;Tachycardia (heart rate> 180 beats per minute for infants and >160 beats per minute for children) or urine output <1 ml/kg/h, if <30 kg and <0.5 ml/kg if >30 kg. 1) Hypovolemic shock was diagnosed when there was history of fluid loss like vomiting, diarrhea, loss of blood etc and physical findings of dehydration and shock. 2) Cardiogenic shock was identified when there was preexisting heart disease or when there were known risk factors to cause myocardial damage like scorpion sting and the findings also pointing towards a primary cardiac involvement and concomitantly having features of shock mentioned above. 3) Septic shock was diagnosed when there was a focus of infection like meningitis, encephalitis, or pneumonia proven by clinical features and appropriate investigations and also having features of hemodynamic compromise. 4) Anaphylactic shock was said to be there when there was sudden cardiovascular collapse following exposure to an inciting agent. Therapy was given based on existing protocols in the institute. For hypovolemic shock fluid boluses were given to restore the blood pressure and then subsequently dehydration assessed and corrected. In children with cardiogenic shock Dobutamine and vasodilator, were used. Septic shock cases were treated with initial 3 boluses of crystalloids and then dopamine started if they had persistent shock. If there was no response to maximum dose of dopamine (15 μg/kg/min), adrenaline infusion was started. In anaphylactic shock cases, adrenaline infusion started along with volume expansion. The outcome measure was ultimate survival or death. Chi-square test has been used to test the significant proportion of study characteristics between two groups. Unpaired t-test was used to compare two means. Binary logistic regression analysis was used to find out independent prognostic factors p value < 0.05 was considered significant The statistical software namely SPSS 11.0. Stata 8.0.Systat 11.0, Medcalc 9.01 and Effect Size calculator were used for the analysis of the data and Microsoft Word and Excel have been used to generate

tables etc. The data was tabulated, analyzed and interpreted

Results

A prospective clinical study of 100 patients with shock was undertaken and above-mentioned clinical and laboratory parameters were compared between survivors and non-survivors. In the present study shows that most common age group is between >1month-5 years, which constitute 77% of total admissions. Out of 100 cases 61% are male and 39% are females. Out of 100 cases 71% survived and 29% were non-survivors. Between > 1month-1year 34.5% are non-survivors and between 1-5 years 48.35% were non-survivors. Out of 29 non-survivors, 16 cases (55.2%) were male and 13 cases (44.8%) were females.(Table no.I) . Septic shock was most common cause of shock 48/100(48%). Followed by hypovolemic 28%, cardiogenic 23% and anaphylactic shock 1%. Septic shock has got highest mortality 65.5%, followed by cardiogenic shock 31%. Least cause of mortality was hypovolemic and anaphylactic shock, which constitutes 3.4% and 0% mortality respectively(Table no. II). Heart rate at admission did not significantly differed between two groups but at 24 and 48 hours it was high in non-survivors than survivors. Systolic blood pressure at admission did not significantly differ between two groups but at 24 hrs and at48 hrs it was significantly low in non- survivors than survivors. Diastolic blood pressure at admission did not significantly differ between two groups but at 24 hrs and at 48 hrs it was significantly low in non-survivors than survivors. At admission mean arterial pressure was higher among non-survivor and after 24 hrs and 48 hrs it declined significantly as compared to MAP among survivor (Table no. III). CFT (sec) at admission was 5.17±1 in survivors and 5.57±1.09 in non-survivors. At 24 hrs of admission it was prolonged in non-survivors (3.34±0.82) as compared to survivors (2.31±0.66) **.**C-PTG (in ^{o}C) was 6.78±2.28 in survivors and 6.96±2.27 in non-survivors. At 24 hrs it was significantly high in non-survivors (6±2.89) as compared to survivors (3.94±2.96). GCS at admission was significantly high in survivors (11.65±2.48) as compared to non-survivors (9.48±1.86). SpO2 at admission was significantly high in survivors (92.28±5.36) as compared to non-survivors (88.21±4.89), $p<0.001$. Urine output at 24 hrs was significantly low in non survivors (0.81±0.34ml/kg/hr) as compared to survivors (1.66±0.55ml/kg/hr). Among investigational parameters, platelet counts were low in non-survivors (1.58±1.31 lac/mm^3) as compared to survivors (2.56±2.07 lac/mm^3), .Creatinine levels were high in non-survivors (1.16±0.47 mg/dL) as compared to survivors (0.92±0.50 mg/dL), PaO_2 was low to non-survivors (72.58±28.6 mmHg) as compared to survivors (105.88±32.72 mmHg), SaO_2 at admission was 92.94±5.18 in survivors and 87.38±7.36 in non-survivors(Table no IV) .

Discussion

Shock is one of the most common emergencies in pediatrics. In our study it is accounted for 100/784(12.7%) admissions in PICU. In a study done by Daljit Singh et al it accounted for 4.5% of PICU admissions [6]. In this study the overall mortality in shock was 29% which is in concordance with that found in the literature (30-60%) [1,7] and Daljith Singh et (26.4%)[6].There was no significant influence of age and sex on the out come in present study. Similar findings have been observed in Daljit Singh et al study[6]. In our study male patients constituted about 61% . This is in accordance to study by Praveen Khilani et al in which males constituted 60% which was mainly due to male dominated society in India[8].In this study septic shock is the most common cause of shock 48%

followed by hypovolemic shock 28% cardiogenic shock accounted for 23% and anaphylactic shock 1%. Though hypovolemic shock is recognized as the most common cause of shock in children, it was not the most common cause of shock in our study 28% [2,3,5,9,10]. Since ours is a tertiary level hospital the complicated cases are referred to our PICU and majority of cases are treated out side in the govt hospitals. Similarly in other study by Chang P et al, it accounted for 7/22(32%) of the cases admitted with shock[7]. The mortality in shock depends on the etiology [3]. In this study septic shock had maximum mortality 65% (19/29) whereas in other studies it ranged from 10-82% in the children[7,11-17]. In a study done by Daljith Singh et al, septic shock has got mortality of 46.7%[6] Cardiogenic shock was found to have mortality of 31% (9/29). In a study by Chang P et al mortality was 75% in cardiogenic shock[7]. Hypovolemic shock had a least mortality in this study 1/29(3.4%), similar to that found in literature 0-20% [7,18,19]. In a study done by Daljit Singh et al mortality due to hypovolemic shock was 2.3%. [6]. acute gastroenteritis was the most common cause of hypovolemic shock in this study as was found in a study by Chang P et al and also according to WHO which states acute diarrhoel disease is one of the most common cause of mortality in children[7,20]. Heart rate (mean±SD, beats/min) at admission was not significantly different in both groups but at 24 hours it was significantly high in non-survivors and returned to normality in survivors . In contrast heart rate did not identify non-survivors from survivors at any time in the first 48 hours according to Duke TD et al in children with septic shock [21]. Changes in the heart rate are an early sign of improvement just as they are an early warning to further deterioration. Though a single cut off cannot be given in children because it varies with age and different causes of shock. In this study, the trend in the heart rate in the first 24 hours after admission did predict survival. The mean arterial pressure (mean±SD, mm of Hg) at 24 hours was significantly higher in survivors (63.51±14.35) than in non-survivors (51.88±7.61) .in this study. Similar finding was noted in the study done by Duke TD et al [21] . C-PTG (mean±SD,^{0}C) was similar at admission (survivors 6.78±2.28, non-survivors 6.96±3.27) at 24 hours after admission it was significantly higher in non-survivors (6.66±2.89) survivors (3.94±2.89)..C-PTG predictedoutcome in this study.CRT (seconds) was significantly prolonged in non-survivors (3.34±0.82) as compared to survivors (2.31±0.66) at 24 hours. In African, meningococcal epidemics, delay in CRT was found to be useful prognostic factor along with other clinical variables. GCG at admission was significantly low in non-survivors (9.48±1.86) than in survivors (11.65±2.48). Similarly in study done by Raicevic R et al, level of consciousness was in positive correlation with outcome, and GCG<8 was an independent predictor of morality in a new prognostic scoring system for meningococcal shock.[22]. SpO_2 (mean±SD, %) was significantly low in non-survivors (88.21±4.89) at admission than in survivors (92.28±5.36) and hence SpO_2 had predictive value. The urine output (mean±ml/kg/hr) at 24 hrs after admission was significantly low in non-survivors (0.81±0.34) than in survivors (1.66±0.55) . Among the investigational parameters studied at admission, decrease in the platelet count is associated with high mortality (non-survivors: 1.58±1.31, survivors: 2.56±2.07). According to Change P et al, thrombocytopenia was an independent risk factor for pediatric shock states[7].There was no significant difference between the urea levels between the survivors (50.10±33.18) and non-survivors (54.61±22.32) in this study.Creatinine level was significantly

high in non-survivors (1.16±0.47) as compared to survivors (0.92±0.50).pH at admission did not predict the survival in this study. In survivors, it was (7.27±0.15), in non-survivors it was 7.20±0.15), . Contrary to this study, Pollack MM et al observed that there were more non survivors than survivors with low pH value[23].In this study PaO_2 at admission (mean±SD) in survivors was (105±32) in non-survivors it was 72.58±28.60 . Similarly, Pollack MM et al noted significantly low PaO_2 in pediatric septic shock cases[23].In this study PCO_2 at admission was significantly high in non-survivors (38.39±21.4 mmHg) as compared to survivors (27.31±9.45)..

Study population (n=100)	Survivors (n=71)		Non-survivors (n=29)		$\chi 2$ value
Age (No)	**No.**	**%**	**No.**	**%**	
Up to 1 years (39)	29	40.8	10	34.5	2.04
1 year -5 years(38)	24	33.8	14	48.3	
5-10 yrs(17)	13	18.3	4	13.8	
>10 yrs(6)	5	7.0	1	3.4	
Sex (No)					
Male(61)	45	63.4	16	55.2	0.583
Female(39)	26	36.6	13	44.8	

Conclusion

Temporal patterns of various clinical parameters showed a trend towards normalization of the various physiological variables in survivors in the first 24-48 hours where as the variables tend to be abnormal in non-survivors.A,t admission GCS , SpO_2 had prognostic value among investigational parameters. Platelet count ,Creatinine levelsPaO_2,SaO_2 has relevance to prognosticate (outcome)

Recommendation

- Continuous hemodynamic monitoring is very important in all cases of shock.
- Early goal directed therapy should be implemented in all cases

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Table

Table I : Distribution of outcome according to age and sex

Table II: Distribution of outcome according to etiology

Study population (n=100)	Survivors (n=71)		Non-survivors (n=29)		$\chi 2$ value	p value
Etiology (No)	**No.**	**%**	**No.**	**%**		
Hypovolemic (28)	27	38.1	1	3.4	12.2	<0.001**
Septic (48)	29	40.8	19	65.5	5.02	0.025*
Cardiogenic (23)	14	19.7	9	31.0	1.49	0.222
Anaphylactic shock (1)	1	1.4	0	-	0.413	0.999

*** moderately significant ** Strongly significant**

Table-III: Comparison of clinical parameters in survivors and non-survivors

clinica	Survivo	Non-	t-	p

l parameters	rs (n=71)	Survivors (n=29)	value	value
Heart rate (bpm)	**Mean± S.D.**	**Mean± S.D.**		
0 hours	164.87± 20.71	156.59± 28.87	1.61	0.111
12 hours	151.31± 21.20	158.34± 17.56	1.58	0.118
24 hours	136.17± 20.08	157.73± 27.34	4.37	<0.001**
48 hours	125.88± 22.74	159.37± 18.43	7.04	<0.001**
SBP (mmHg)				
0 hours	69.34±9 .24	70.46±1 8.80	0.40	0.065 +
12 hours	78.30±1 5.84	73.79±1 2.46	1.37	0.180
24 hours	87.55±1 6.25	71.93±1 1.49	4.71	<0.001**
48 hours	90.93±1 6.53	71.37±6 .81	6.15	<0.001**
DBP (mmHg)				
O hours	39.59±1 2.83	49.10±1 5.47	3.06	0.066
12 hours	43.69±1 5.67	39.19±1 0.32	1.42	0.170
24 hours	51.49±1 3.99	40.32±6 .81	4.09	0.001 **
48 hours	54.64±1 0.88	35.95±1 0.42	6.62	<0.001**
MAP (mmHg)				
O hours	50.37±1 0.69	60.96±1 6.26	3.83	0.031 *
12 hours	55.23±1 5.19	50.89±1 0.68	1.40	0.177
24 hours	63.51±1 4.35	51.88±7 .61	4.13	0.001 **
48 hours	66.74±1 1.29	47.75±8 .70	8.12	<0.001**

*** moderately significant ** Strongly significant**

Table IV: Comparison of study parameters in survivors and non-survivors

Parameters	Survivors (n=71)		Non-survivors (n=29)		t-value	p values
	Mean	SD	Mean	SD		
Capillary Refilling time at admission	5.17	1.0	5.57	1.09	1.77	0.103
Capillary Refilling time at 24 hrs	2.31	0.66	3.44	0.82	7.23	0.001**
Core-Peripheral temp gradient- ^{o}C at admission	6.78	2.28	6.96	2.27	1.15	0.252
Core-Perip	3.94	2.96	6.0	2.89	3.18	0.003**

heral temp gradient at 24 hrs						
GCS at admission	11.65	2.48	9.48	1.86	4.24	<0.001**
SpO_2 at admission	92.28	5.36	88.21	4.89	3.70	0.001**
Urine output at 24 hours	1.66	0.55	0.81	0.34	7.73	<0.001**
Hemoglobin	9.57	2.71	8.83	1.85	1.35	0.189
Total count	13213.80	7360.38	14660.71	10990.78	0.77	0.449
Band cell	7.17	3.91	13.18	7.63	6.19	<0.001**
Platelet	2.56	2.07	1.58	1.31	2.36	0.022*
Na	136.01	7.70	133.71	11.41	1.17	0.249
K	4.62	1.34	4.52	0.96	0.36	0.728
CI	104.72	8.51	104.96	5.69	0.14	0.888
Urea	50.10	33.18	54.61	22.32	0.67	0.510
Creatinine	0.92	0.50	1.16	0.47	2.21	0.027*
Ph	7.27	0.15	7.20	0.15	2.12	0.047*
PaO_2	105.88	32.72	72.58	28.60	4.78	<0.001**
PCO_2	27.31	9.45	38.39	21.40	3.60	0.001**
HCO_3	13.79	5.60	14.73	4.31	0.81	0.426
SaO_2	92.94	5.18	87.38	7.36	4.29	<0.001**
SGOT	104.36	128.95	97.17	60.27	0.29	0.779
SGPT	104.96	107.48	101.81	68.28	0.15	0.887
Calcium	7.88	1.17	7.56	1.16	1.24	0.212
Phosphorus	4.38	1.03	4.50	0.66	0.58	0.564

*** moderately significant ** Strongly significant**

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